Trigeminal Neuralgia
Diagnosis and Treatment

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KEYWORDS

• Facial pain • Neuropathic pain • Trigeminal neuralgia • Trigeminal nerve
• Surgical treatment • Pharmacologic management

KEY POINTS

- Trigeminal neuralgia is frequently mis-/underdiagnosed in the population.
- Pharmacologic management of trigeminal neuralgia remains the first-line treatment.
- The gold standard for the surgical treatment of classic trigeminal neuralgia secondary to neurovascular impingement is microvascular decompression.
- Percutaneous modalities offer additional treatment options for secondary causes of trigeminal neuralgia.
- Stereotactic radiosurgery offers a favorable pain relief for patients without vascular compression or who would like to avoid craniotomy.

INTRODUCTION

Trigeminal neuralgia (TN), the most common craniofacial neuralgia, results in intense, debilitating facial pain and can profoundly affect quality of life. In this article, the authors summarize the characteristics, pathophysiology, and management of TN in both primary and secondary care settings.

Definition and Classification

TN classically presents with "recurrent unilateral brief electric shocklike pains, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli."[1] More recently, it has been understood that up to 30% of patients may present with continuous pain of moderate

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intensity within the same distribution as the affected nerve division.[1] Various classification methods have been proposed for facial pain. The International Headache Society has divided TN into 3 main categories: classic, secondary/symptomatic, and idiopathic.[1] Classic TN is associated with neurovascular compression (NVC) of the trigeminal nerve root with associated morphologic changes on MRI.[1] Secondary/symptomatic trigeminal (STN) neuralgia is associated with an underlying disease. The most common causes include multiple sclerosis (MS), space-occupying lesions, skull-base bone deformity, connective tissue diseases, arteriovenous malformations, dural arteriovenous fistulae, or genetic causes of neuropathy.[1] Idiopathic TN (ITN) is a diagnosis of exclusion, when neither classic nor secondary TN is identified through appropriate testing (eg, MRI and electrophysiological tests).[1] Both classic TN and ITN can further be classified based on the presence of concomitant pain.

Symptomology

TN is usually unilateral with an increased prevalence of right-sided pain.[1–5] The few cases in which TN presents bilaterally are often secondary TN, and pain in one of the sides exhibits temporal separation from the other side.[5–8] The location of pain in TN is divided based on the 3 distributions of the trigeminal nerve: V1 (ophthalmic), V2 (maxillary), and V3 (mandibular). Approximately 36% to 42% of patients exhibit pain in one distribution alone, with V2/V3 representing the most common distributions. In 35% of patients, pain is found in both V2 and V3 distributions with 14% of patients experiencing pain in all 3 distributions.[2,4,5,9–13] Given these distributions, patients frequently initially present to dentists with tooth pain. Another characteristic of TN is the evocable nature of the pain. Common daily activities (eg, chewing, talking, shaving, brushing teeth, and light touch) in the affected distribution can trigger TN pain.[1,3,4] Although patients may report spontaneous pain, it is debated whether the pain is truly spontaneous or if subtle sensory stimulation outside of conscious awareness triggers the attack. Pain is often followed by refractory period in which a new attack cannot be elicited. The pathophysiology of the refractory period is poorly understood, but the intensity and duration of an attack seems to correlate with the duration of the refractory period.[14] TN does not frequently present with sensory deficits on gross examination; however, advanced sensory testing (ie, quantitative sensory testing) can elicit subtle findings.[15,16] Indeed, a recent large series identified sensory abnormalities in 30% of patients. Autonomic symptoms were traditionally not considered a part of TN; however, recent work has shown that autonomic symptoms, in particular lacrimation and rhinorrhea, can occur in a large proportion of patients with TN.[2,4,17–21] The presence of autonomic symptoms can make the diagnosis of TN challenging, given overlap between short-lasting unilateral neuralgiform headaches with autonomic signs (SUNHA).[5,20,21] In fact, elucidating between SUNHA and TN with lacrimation can be so difficult that the patient may be assigned both diagnoses initially.[1,5]

Pathophysiology

Despite variable TN causes, the prevailing pathophysiologic theory remains similar. ITN results in a focal demyelination whose cause is unknown, whereas primary demyelination or demyelination secondary as a result of external insults has been linked to the hyperexcitability of primary afferents, leading to several downstream effects at the neuronal level that is thought to be responsible for symptom onset.[22] Animal models and patient biopsies suggest a cause to be related to the dysregulation of voltage-gated sodium channels (Na_v).[22,23] Specifically, both the $Na_v1.3$ and $Na_v1.1$ channels are upregulated, whereas $Na_v1.7$ is downregulated.[22,24–26]

Advancements in the histopathologic and electrophysiologic understanding of TN led Devor and colleagues, in 2002, to propose the original theory for the cause of symptoms of TN, which was termed the "ignition hypothesis."[27] Electrophysiological recordings obtained from the root entry zone of the trigeminal nerve demonstrated an ectopic generation of action potentials.[22,28–31] Dysfunctional ephaptic transmissions of the various types of demyelinated neurons was thought to be the result of a collective summation of the up-and-down regulation among the aforementioned sodium channels. It was this dysfunctional sensory transmission that was thought to be the source for the propagation of numerous amplified ectopic action potentials and the overall functional hyperexcitability responsible for the characteristic shocklike symptoms patients experienced.[22,32–34]

Numerous studies have since pointed to the demyelination of primary trigeminal afferents near the entry of the trigeminal root into the pons (dorsal root entry zone) as the pathophysiological mechanism of TN.[19,35–37] In particular, the transition from oligodendrocyte to Schwann cell myelination within the proximal 25% of the trigeminal nerve is particularly susceptible to insult representing a *locus minoris resistentiae*.[38] Furthermore, the hyperexcitability of afferent trigeminal nerves causes the sensitization of multiple areas within the central nervous system, including the spinal trigeminal nucleus, thalamus, and somatosensory cortices, leading to the further perpetuation of pain.[22,39,40]

In classic TN, it has been most commonly attributed to NVC by the superior cerebellar artery, located within the cerebellopontine cistern, which is thought to result in focal demyelination and hyperexcitability of the involved nerve.[19,41,42] Although in some cases, simply the mere contact of the artery to the nerve, even in the absence of morphologic changes, can lead to symptoms.[43,44] Burchiel and colleagues found that the association of classic TN with NVC was insignificant, as 99.94% of compression in the general population was asymptomatic. In addition, patients aged 20 to 40 years who presented with classic TN were much less likely to have evidence of NVC than patients older than 50 years.[45] Thus neither the presence nor absence of NVC is sufficient to diagnose TN; the patient must be symptomatic. Patients with TN can go for years at a time without a recurrence of pain between episodes. It has been suggested that a partial remyelination of the affected neurons may be attributable to this phenomenon as well as the spontaneous remission of pain described in some patients.[32]

In secondary TN, demyelination occurs because of other causes such as MS or space-occupying lesions. More specifically, in patients with multiple sclerosis, intrapontine demyelination can be an additional cause for the TN symptom onset.[5,19] In some cases, patients may present with a demyelinating plaque in addition to NVC at the root entry zone, representing a "double-crush" mechanism. Most of the patients undergoing a microvascular decompression have been shown to have severe NVC intraoperatively. However, in these patients, microvascular decompression is generally reported to be less effective.[46]

Trigeminal Neuralgia in Multiple Sclerosis

Compared with the general population, patients with MS have a 20-fold increased risk of developing TN. Typically, the pain symptoms associated with TN become apparent 12 years after the initial onset of MS symptoms.[47–49] MS-associated secondary TN differs significantly in regard to classic TN. In general, it often presents at a younger age and more commonly, occurs in the trigeminal distribution bilaterally.[50] As a result, the treatment pathway for the associated secondary TN symptoms in MS differs from those with the classic form. Patients tend to undergo surgical interventions earlier,

as pharmacologic options can further exacerbate MS symptoms.[51] In addition, patients also tend to have an increased number and more frequent surgeries than patients with classic TN.[51]

DIAGNOSIS OF TRIGEMINAL NEURALGIA

A proper and thorough patient history is of the utmost importance when diagnosing TN, as it is a partially exclusionary diagnosis that is based on specific clinical features, whereas subsequent testing may be used to further elucidate the specific cause.[1]

The third edition of the International Classification of Headache Disorders (ICHD-3) by the International Headache Society, published in 2018, defined the features necessary for the diagnosis of TN.

Diagnostic Criteria

A. Recurrent unilateral facial pain in one or more distributions of the trigeminal nerve with no radiation beyond and fulfilling criteria B and C
B. Pain has the following characteristics:
   1. Lasts from a fraction of a second to 2 minutes
   2. Severe intensity
   3. Electric shocklike, shooting, stabbing, or sharp in quality
C. Precipitated by innocuous stimuli within the affected trigeminal distribution
D. Is not accounted for by another ICHD-3 diagnosis[1]

A diagnosis of classic TN fulfills the aforementioned criteria and demonstrates NVC of the trigeminal nerve root with associated morphologic changes seen either on preoperative MRI or by direct visualization intraoperatively.

Classic TN can further be broken down into classic TN with purely paroxysmal pain in which the interval between attacks is pain free and classic TN with concomitant continuous pain in which there is continuous or near-continuous pain between attacks in the affected distribution.

Secondary TN is diagnosed based on the fulfillment of the criteria for TN highlighted earlier along with the pain attributed to a separate causative comorbid condition. Within secondary TN, there are 3 subdiagnoses: TN attributed to MS, TN attributed to space-occupying lesions, and TN attributed to other causes.

Finally, ITN is diagnosed when the criterion for TN is met, but neither classic nor secondary TN can be diagnosed based on MRI and electrophysiological tests. Similarly, to classic TN, idiopathic TN can be subdivided into purely paroxysmal pain or TN with concomitant continuous pain.

TN is frequently both mis- and under-diagnosed in the primary and secondary care settings. Although clear diagnostic criteria exist, the variable presentation of symptoms as well as their distribution and sometimes subtle pain qualities can lead to misdiagnosis in various clinical environments. Patients with TN consult a variety of medical professionals for their pain including primary care physicians, dentists, otolaryngologists, neurosurgeons, neurologists and/or headache specialists.[5,52] It is important to consider as well as exclude alternative diagnoses before arriving at TN as the final diagnosis.

The differential diagnoses for TN pain are expansive. The following list, although not complete, lists those that should be considered most often: glossopharyngeal neuralgia, painful posttraumatic trigeminal neuropathy, persistent idiopathic facial pain, painful trigeminal neuropathy attributed to acute herpes zoster, burning mouth syndrome, short-lasting unilateral neuralgiform headache attacks with autonomic symptoms, short-lasting unilateral neuralgiform headache attacks with conjunctival

injection and tearing, migraine variants, paroxysmal hemicrania, cluster headaches, giant cell arteritis, occipital neuralgia, nervus intermedius neuralgia, perineural head and neck malignancies, trigeminal nerve or tract lesions, cavernous or cerebellopontine angle lesion, dental pain, temporomandibular joint dysfunction, osteomyelitis, glaucoma, sinus disease, and otitis media.[5,19,52,53] A careful patient history should provide particular characteristics of the location, onset, character, and duration of pain. Further questioning should seek to identify any associated symptoms in order to rule out alternative diagnoses (**Table 1**) before the diagnosis of TN.

MANAGEMENT AND TREATMENT OF TRIGEMINAL NEURALGIA

Pharmacologic agents are considered first line for the treatment of TN regardless of cause. Surgical or interventional procedure modalities are considered only when either pharmacologic measures are contraindicated or when the pain becomes refractory to medication therapy. Postoperatively, patients will often continue medical therapy albeit at lower doses.[42]

Pharmacologic Management Options

Before treatment, electrocardiogram and laboratory testing are done to ensure proper heart, liver, and kidney function. Furthermore, most medications used to treat TN pain are teratogenic. Women who expect to become pregnant or are of child-bearing age should be appropriately counseled. There are currently 10 Food and Drug Administration–approved medications for the treatment of TN: carbamazepine, oxcarbazepine, baclofen, lamotrigine, pimozide, gabapentin ± ropivacaine, phenytoin, tizanidine, and botulinum toxin A. In addition, pregabalin, topiramate, levetiracetam, and vixotrigine, according to various case series and reports, suggest some treatment response but their use remains off-label.

Carbamazepine/Oxcarbazepine

Carbamazepine is a sodium channel blocker with level A evidence for its use and is the most commonly prescribed first-line medication for the management of symptoms associated with TN.[54–58] Initial dosage is generally 100 to 200 mg twice a day, and escalations are done gradually (100 mg every other day up to 1600 mg) to avoid adverse reactions until pain relieved or side effects occur.[59] The number needed to treat (NNT) for any pain relief is 1.9 and 2.6 for significant pain relief,[5,60,61] whereas the number needed to harm is 3.4 for minor reactions and 24 for serious events.[55,59]

Among severe adverse events, Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) have been shown to be associated with individuals who harbor the HLA-B*1502 allele when taking carbamazepine and is more commonly found in certain Asian populations. Carbamazepine is contraindicated in patients with atrioventricular conduction abnormalities and before its use all patients should undergo an electrocardiogram. It can also result in several other conditions including hyponatremia, certain blood disorders, and multiple neurologic side effects (eg, tiredness, sleepiness, memory problems, disturbed sleep, difficulty concentrating, and unsteadiness).[59,62] Routine blood sampling is necessary to monitor cell count and organ function.

Oxcarbazepine is a derivative of carbamazepine and has also been shown to be effective in reducing pain from TN. Daily dose ranges are between 300 and 1200 mg.[59] Although oxcarbazepine has the benefit of having far fewer side effects, when compared with carbamazepine, the body of evidence is lacking (level B evidence) and as a result is generally considered a second-line treatment option.[54,55,62]

Table 1
List of alternative diagnoses for trigeminal neuralgia sorted by characteristic features

Characteristic of Pain	Distinguishing Features	Alternative Diagnoses
Onset	Recent rash related to herpes zoster infection	Painful trigeminal neuropathy attributed to acute herpes zoster
	Injury or accident	Painful posttraumatic trigeminal neuropathy
Location	Pain in the jaw or teeth	Cracked tooth or carries/pulpitis
	Pain in the scalp or occipital region	Occipital neuralgia, primary stabbing headaches, or paroxysmal hemicrania
	Pain in the back of the tongue or soft palate	Glossopharyngeal neuralgia
	Pain deep in the ear	Nervus intermedius neuralgia
	Bilateral pain	Tension-type headaches, temporomandibular joint disorder, or persistent idiopathic facial pain
Duration	Constant pain	Otitis, giant cell arteritis, osteomyelitis, burning mouth syndrome, or trigeminal neuropathy
Associated symptoms	Autonomic symptoms	SUNA, SUNCT, or paroxysmal hemicrania

Recent pharmacodynamic and pharmacokinetic studies for these medications have shown gender-specific differences that become important and should be considered when determining the rate and extent of dose escalations. Women demonstrate adverse effects at significantly lower doses. The potential toxic daily dose in women was 800 mg and 1200 mg of carbamazepine and oxcarbazepine, respectively. Conversely, the toxic daily dose for men was 1200 mg and 1800 mg of carbamazepine and oxcarbazepine, respectively.[5,63]

Other Medications

Baclofen, lamotrigine, gabapentin ± ropivacaine, pimozide, phenytoin, tizanidine, and botulinum toxin A are all supported by randomized controlled trials (level C evidence). However, they are used most commonly as therapeutic adjunct or after first- and second-line medications have failed.[54,55]

Baclofen is a γ-aminobutyric acid type B receptor agonist with a relatively safe side-effect profile. The NTT is 1.4, with most patients requiring 50 to 80 mg daily. Because of its mechanism of action, it important to wean off the drug slowly to avoid withdrawal-like symptoms. Baclofen demonstrates synergistic effects with carbamazepine and is regularly used in combination therapy.[64,65]

Lamotrigine is another second-line medication, with daily dosing between 200 and 400 mg and can be used in combination with carbamazepine.[59,66,67] Similar to carbamazepine and oxcarbazepine, lamotrigine requires gradual dose escalation to avoid SJS, TEN, and DRESS syndrome.

Gabapentin exerts its effects on voltage-activated calcium channels, limiting neurotransmitter release and has been shown to significantly reduce neuropathic pain in

multiple randomized controlled trials. But there has been only one study that has specifically investigated the use of gabapentin, albeit in conjunction with ropivacaine, for TN.[68] Gabapentin has several advantages favoring its use: faster dose titration ability and a favorable side-effect profile with limited concern for severe drug or adverse skin reactions seen with the other medications. It is generally also used in combination therapy with other drugs.

Pimozide is a dopamine receptor antagonist that has been tested in a randomized double-blind crossover trial for refractory TN. Despite excellent pain control outcomes, it is rarely used due to its severe side-effect profile that includes risk of arrhythmias, extrapyramidal symptoms, and Parkinsonism.[69]

Phenytoin, another sodium channel blocker, is generally used adjunctively with carbamazepine with daily dose ranges between 200 and 300 mg. It has a large side-effect profile, and its routine use for chronic symptom management has fallen out of favor. However, it remains effective option in acute settings because intravenous boluses allow for rapid therapeutic serum levels.

Tizanidine is an alpha-adrenergic agonist with limited evidence to support its use. In a small trial all treated patients (n = 10) who initially had good pain relief developed recurrence of symptoms within 3 months.[70]

Botulinum toxin A (BTA) has been useful for the treatment of other pain conditions such as migraines, tension headaches, occipital headaches, and postherpetic neuralgias. To date, there has been one randomized controlled trial and 5 prospective open-label studies that have investigated the use of BTA for the treatment of TN.[71–76] In these studies, patients were injected between 5 and 75 units. Most of the patients received a dose of between 20 and 50 units.[5] Four weeks postprocedures, pain relief was reported in 70% to 100% of all patients, and there was a 60% to 100% reduction in the mean pain intensity and frequency. In another study following injection of BTA, 47% of patients did not require any further treatment, 33% of patients continued use of nonsteroidal antiinflammatory drugs, and the remaining 20% had good response with the use of traditional pharmacologic therapy.[73] Initial studies have yield significant pain relief outcomes; however, further investigation is necessary to better understand its indications as well as any long-term recurrence and complication rates.

Some medications have shown some evidence for use in small, lower evidence studies. In an open-label study, pregabalin, a medication structurally similar to gabapentin, showed a reduction in TN-associated pain in 50% to 74% of patients.[77] Similarly, a small study showed pain reduction in 75% taking topiramate for symptoms of classic TN.[78] But when carbamazepine alone was compared with the combination therapy of carbamazepine and topiramate, there was no additional benefit.[79] Levetiracetam is a newer antiepileptic medication that inhibits presynaptic calcium channels thereby decreasing neurotransmitter release. In 2 small studies investigating its use, patients reported 50% to 90% improvement in pain and a 62% reduction in daily attacks.[80,81] Lastly, vixotrigine is a $Na_v1.7$ selective sodium channel blocker that is currently undergoing a phase III randomized controlled trial.[82] Preliminary evidence from a phase 2a withdrawal randomized controlled trial indicated that vixotrigine had a moderate effect.[83]

Surgical and Radiosurgical Treatment Options

Although there are no specific guidelines for the selection and timing, it is recommended that patients be managed as part of a multidisciplinary team. In general, surgical options should be considered after the patient has either failure or had an adequate trial of medication management. In addition, the patient's age, general health,

surgeon's level of expertise, and the available facilities may further influence the consideration and/or timing of any surgical intervention.

Percutaneous Treatment Options

Percutaneous procedures seek to create a focal injury to trigeminal nerve afferents within the gasserian ganglion. These procedures include the most commonly performed thermocoagulation via radiofrequency ablation, as well as chemical neurolysis via glycerol injection, mechanical neurolysis via balloon compression nerve blocks, and cryotherapy. Historically these procedures have been used most in patients older than 65 years, as well as patients with MS, pontine infarct, or local mass lesions affecting the dorsal root entry zone.

Radiofrequency (RF) ablation is performed typically under short-acting general anesthesia or deep sedation, whereas a cannula and RF probe are inserted into the trigeminal ganglion through foramen ovale. The entry point is 2.5 cm lateral to the corner of the mouth and 1 cm inferior with a trajectory aimed toward a point 3 cm anterior to the ipsilateral external auditory meatus and in the plane of the midpupillary line. Glycopyrrolate may be give before entering the foramen ovale with the cannula to avoid the vagal effects that can occur with dural puncture. The initial placement of the probe is determined based on the dermatomal distribution of pain as well as the relationship of the cannula to the clival line with the use of intraoperative fluoroscopy. The patient is then partially awoken to confirm proper placement with the use of sensory test stimulation (100 Hz, 1 msec pulse width, 0.1–0.15 V amplitude). Afterward the patient is placed back under general anesthesia for the ablation. The ablation typically consists of 2 lesions made at 70 to 80°C for 90 seconds each. The use of sensory test stimulation can help improve the selectivity of the procedure. Nearly 100% of patients with an adequate ablation will experience immediate pain relief. It is expected that patients have some numbness within the prior pain distribution area.

The technique for glycerol rhizotomy is similar to RF ablation in terms of obtaining access to the trigeminal cistern. Once access is obtained, the patient is repositioned to a sitting position with their head flexed. A cisternogram is then performed to estimate the volume of the trigeminal cistern. Afterward glycerol is slowly injected to the appropriate volume, and the patient remains upright, sitting with their head flexed for at least 1 to 2 hours. The effect is not immediate, and it can in some instances take up to 2 weeks for pain relief.[84]

Balloon compression does not require the patient to be awake and can be done under general anesthesia. The foramen ovale is similarly accessed as previously described, at which point a No. 4 Fogarty balloon catheter is placed through the cannula and inflated to an intraluminal pressure of 1200 to 1500 mm Hg and maintained for 1 to 2 minutes; this results in compression of the nerve with presumed sparing of the small unmyelinated fibers responsible for corneal reflex and is thought to provide additionally an aspect of dural stretching, resulting in some *decompression* of the trigeminal ganglion. Most patients experience pain relief immediately or within 1 to 2 days following the procedure.

A pooled analysis showed that 19% to 58% of patients were pain free at 4 to 11 years after glycerol injection, 26% to 82% after radiofrequency ablation, and 55% to 80% after balloon compression.[85] Although pain-free rates are promising, large variations between institutions prevent strong conclusions from being drawn. Multiple treatments can improve outcomes but at the risk of increased morbidity, most notably dysesthesias and anesthesia dolorosa.

Nerve blocks can be helpful in an acute setting. Their effect typically lasts only hours but can be helpful in managing severe exacerbations, allowing time for other administered medications to start working.[86,87]

Microvascular Decompression

Microvascular decompression remains the gold-standard primary surgical treatment of patients suffering from classic TN symptoms caused by neurovascular conflict. For secondary TN that remains refractory to pharmacologic management or treatment of the underlying cause and for idiopathic TN any of the percutaneous ablative procedure of the gasserian ganglion described earlier may be performed.

The goal of microvascular decompression (MVD) is to separate the trigeminal nerve root from the blood vessels that are in contact with the nerve root; this is done through a retrosigmoid craniotomy to gain access to the trigeminal nerve root within the posterior cranial fossa. Although this procedure is the most invasive technique for the treatment of TN, it has been shown to provide the lowest rate of pain recurrence and the highest patient satisfaction.[88] Greater than 73% of patients maintained pain relief 5 years after the procedure, with 62% to 89% of patients with classic TN pain free at 3 to 11 years.[54,55,85] In general, the average TN recurrence rate is approximately 4% per year.[89]

Complications from an MVD are rare, and morbidity ranges from 0.3% to 3% and mortality from 0.2% to 0.5%. The lowest complications are seen at high-volume hospital centers where the procedure is done routinely.[90] Complications can include cerebral spinal fluid leak, aseptic meningitis, cerebral infarcts, hematomas, unilateral hearing loss, facial weakness, and facial sensory loss.[54,55,91]

Stereotactic Radiosurgery

Stereotactic radiosurgery is another ablative procedure that can help treat TN. One common modality, Gamma Knife, uses radiation (75–85 Gy) from a cobalt-60 gamma emission source to target the root entry zone of the trigeminal nerve root and can be used in patients with or without vascular compression. Because of the destructive nature of radiosurgery, patients often endorse sensory loss following the procedure. Higher radiation doses resulted in better pain relief outcomes but come at the expense of an increase in the area of sensory loss.[54,55] Pooled analysis demonstrates that 30% to 66% of patients remained pain free 4 to 11 years after surgery.[85] Because of mechanistic action of radiation therapy, pain relief on average is delayed 1 month. Other studies have shown that the proportion of patients who have pain relief can continue to increase for up to 24 months after radiation.[5,92–94]

Although microvascular decompression has been shown to have longer period of pain relief and lower rates of recurrence (70%–74% relief at 10 years; 18% recurrence at 25 years) compared with Gamma Knife Radiosurgery (GKRS) (67% relief at 3 years), the less-invasive nature of radiosurgery allows it to be offered to patients who may be ineligible for the more invasive microvascular decompression while still offering favorable pain relief.[95,96]

SUMMARY

In recent years, the classic definitions for TN have been revised as a result of improved mechanistic understanding of the pathophysiology. Initially, the pain management and treatment of TN begins with an accurate diagnosis among myriad of differential diagnoses, followed by an adequate trial of one of the many effective pharmacologic options currently available. Percutaneous and open microvascular surgical

decompression or radiosurgery of the trigeminal nerve are often reserved for those patients who continue to have symptoms despite a period of nonsurgical management. Surgical interventions must be individually tailored in terms of the type of TN present and taking into consideration the treatment goals of the patient. Despite the volume of research currently available of this topic, there is still a need for continued research into the pathophysiology and management of TN.

CLINICS CARE POINTS

- It is important to consider all alternative diagnoses and exclude them before diagnosing a patient with TN.
- Carbamazepine is a first-line treatment of TN with level A evidence supporting its use.
- Surgical treatments of TN are considered when pain is refractory to medications or medications are not indicated.

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